SFUND RECORDS CTR 101656

CONTRACTOR OF THE ONLY THE EXPLOSIVE CONTRACTOR OF THE CONTRACTOR

OPERATED BY UNION CARBIDE CORPORATION FOR THE ENERGY RESEARCH AND DEVELOPMENT ADMINISTRATION

NOTICE

ility,

RTIONS OF THIS FEPORT ARE ILLEGIBLE. It is been reproduced from the best evailable by to permit the breadest possible available.

ORNL/TIRC-76/2

TRICHLOROETHYLENE

I. An Impact Overview

E. M. Waters - H. B. Gerstner - J. E. Huff

II. An Abstracted Literature Collection 1907 - 1976

E. M. Waters - S. A. Black

MAY 1976

NATIONAL LIBRARY OF MEDICINE TOXICOLOGY INFORMATION PROGRAM

TOXICOLOGY INFORMATION RESPONSE CENTER

ABSTRACT

Trichloroethylene (TCE) has been an industrial chemical of some importance for the past 50 years. First synthesized by Fischer in 1864, TCE has enjoyed considerable industrial usage as a degreaser and limited medical use as an inhalation anesthetic and analgesic.

This TCE overview provides a narrative survey of the reference literature. Highlights include history, nomenclature, physical and chemical properties, manufacture, analysis, uses, metabolism, toxicology, carcinogenic potential, exposure routes, recommended standards, and conclusions.

Chemically, TCE is a colorless, highly volatile liquid of molecular formula C_2HCl_3 . Autoxidation of the unstable compound yields acidic products. Stabilizers are added to retard decomposition. TCE's multitude of industrial uses center around its highly effective fat-solvent properties.

Metabolically, TCE is transformed in the liver to trichloroacetic acid, trichloroethanol, and trichloroethanol glucuronide; these breakdown products are excreted through the kidneys.

Most toxic responses occur as a result of industrial exposures. TCE affects principally the central nervous system (CNS). Short exposures result in subjective symptoms such as headache, nausea, and incoordination. Longer exposures may result in CNS depression, hepatorenal failure, and increased cardiac output. Cases of sudden death following TCE exposure are generally attributed to ventricular fibrillation. Current interest about TCE has focused on recent experimental data which implicate TCE as a cause of hepatocellular carcinoma in mice. No epidemiological data are available which demonstrate a similar action in man. The overall population is exposed to TCE through household cleaning fluids, decaffeinated coffee, and some spice extracts.

The NIOSH criteria standard for TCE stands at 100 ppm as a timeweighted average for an eight-hour day, with a maximum allowable peak concentration of 150 ppm for 10 minutes.

TABLE 2

HISTORY OF TRICHLOROETHYLENE

1864	First prepared by Fischer
1906	First patent held by Konsortium fur Elektrochemische Industrie, Nurnberg
1911	Narcotic properties discovered by Lehman
1914 - 1918	Limited use as a degreaser and solvent
1915	Trigeminal analgesia reported by Plessner
1920's	More widespread use in metal degreasing
1930's	Use spreads to dry cleaning industry
1933	Jackson successfully anesthetizes dogs
1940's	Use in Great Britain as inexpensive, non-explosive anesthetic
1945	Use as anesthetic spreads to USA, does not gain widespread popularity
1960's	"Carbona®-cult" solvent-sniffing
1966	Use as a solvent curtailed in Los Angeles County, CA as a result of evidence implicating TCE in severe smog formation
1975	Preliminary report indicating carcinogenicity
1975	Invocation of Delaney Clause sought to ban all uses in foods

4

Potentiometry (Deyl and Effenberger, 1958) and nephelometry (Effenberger and Deyl, 1958) are accepted methods for determining TCE in waste waters.

te land emiliant entre carrella de la company de la compan

TCE may be assayed colorimetrically in almost any medium using a modified Fujiwara test -- a nonspecific color reaction for highly chlorinated compounds. Procedurally, solid potassium hydroxide, pyridine, and water are heated; the material to be assayed, either urine or an ether extract of blood or tissues is added and the mixture reheated. A red color indicates the presence of TCE. Sunshine (1969) assigns this test a sensitivity of 1 ppm.

USES

Trichloroethylene is used as an industrial solvent, as a household cleaner and solvent, and as an inhalation analgesic and anesthetic.

Medical

TCE has been employed in anthelmintic preparations and for bacteriocidal and fungicidal purposes. During the 1940's TCE enjoyed considerable popularity as an analgesic and anesthetic, particularly in obstetrics; however, this short-lived popularity declined due to the subsequent synthesis of more effective and versatile halogenated anesthetic agents.

Anesthetic. The use of TCE as an anesthetic has largely been abandoned because it does not produce sufficient skeletal muscle relaxation necessary for many surgical procedures. To determine TCE use statistics in hospitals with more than 100 beds, NIOSH surveyed 1,254 hospitals and found that 63 (or 5 percent) used TCE (Medical World News, July 28, 1975). Another source (Chem. Eng. News, May 19, 1975) quotes a maximum figure of 60,000 patients a year receiving TCE during anesthetic procedures.

Induction and recovery are slow due to the high solubility of this compound in blood (Aviado, 1972). TCE is believed to stimulate the pulmonary stretch receptors governing lung deflation, probably resulting in the shallow, accelerated breathing which is characteristic of TCE anesthesia (Whitteridge and Bulbring, 1944). TCE causes a dose-dependent depression of cardiac contractility; spontaneous cardiac arrhythmias have been reported but the experimental evidence is contradictory (Aviado, 1972). TCE is reported to sensitize the heart to catecholamines, particularly epinephrine, resulting in ventricular fibrillation. TCE still is used for short operative procedures in obstetrics, dentistry, burn dressing, and cystoscopy.

Many of the early poisoning incidents during TCE anesthesia resulted from using a closed-system rebreathing apparatus where soda lime acted as a carbon dioxide absorber; however, later investigations revealed that passage of TCE over soda lime results in the production of dichloroacetylene, a neurotoxic agent.

TCE is contraindicated in anemia, toxemia of pregnancy, and diseases of the heart, lungs, and kidneys. TCE is not advocated for use in children or together with epinephrine because of the high risk of ventricular fibrillation (Osol and Pratt, 1973).

Analgesic. Plessner (1915) reported trigeminal desensitization following TCE anesthesia and for a short time TCE use was advocated in the treatment of trigeminal neuralgia. The analgesic properties are attributed to a defatting action on the myelin nerve sheaths.

Obstetrics. As an analgesic for women in labor, TCE is suitable where less than 10 minutes of light anesthesia is needed. Self-administration for obstetrical analgesia has proved to be effective and safe.

The availability of pure TCE preparations and the development of special inhaler devices have enhanced the use of vapors for obstetrical analgesia; however, the onset of analgesia is slow and TCE is potentially cardiotoxic and hepatotoxic (Swinyard, 1975).

Dentistry. For dental extractions, incision of furuncles, and other short operative procedures, TCE has the advantage of being a satisfactory analgesic that is readily portable, inexpensive, and not unpleasant to inhale, coupled with a wide margin of safety between analgesic effect and toxic doses. Excessive salivation, however, may be troublesome in dental procedures (Osol and Pratt, 1973).

Veterinary medicine. TCE is used as an inhalation anesthetic for animals such as pigs, dogs, and cats; as a disinfectant and detergent for skin, minor wounds, and surgical instruments; and as a solvent for the removal of grease from fur, hair, and wounds of animals (Stetcher, 1968).

Agriculture

TCE has been cancelled for use in fumigant mixtures or as a solvent with other ingredients on grains (Farm Chemicals Handbook, 1976).

Food Production

TCE is used in the decaffeination of coffee and the extraction of spice oleoresins. Under Section 121.1041, the Food & Drug Administration has established tolerances of 25 ppm for TCE in decaffeinated ground coffee, 10 ppm in decaffeinated instant coffee, and 30 ppm in spice oleoresins (Cc/de of Federal Regulations, Title 21, 1974).

61

Nakaaki, K., Onishi, N., Hiroyasu, I., Kimotsuki, K., Fukaberi, S. 1973. Experimental study on the effect of exposure to trichloroethylene in map. Rodo Kagaku 49(8):499-563.

National Cancer Institute. 1976. <u>Carcinogenesis bioassay of trichloroethylene</u>. NCI Carcinogenesis <u>Technical Report Series</u> Number 2, NCI-CG-TR-2, DHEW Publication No. (NIH) 76-802. 197 pp.

National Clearinghouse for Poison Control Centers. 1967. Trichloroethylene Congener Card. No. 82. DHEW-PHS.

NIOSH. 1973. Criteria for a recommended standard: occupational exposure to trichloroethylene. DHEW/PHS. Washington: U.S. GPO.

Nomura, S. 1962. Health hazards in workers exposed to trichloroethylene vapor. I. Trichloroethylene poisoning in an electroplating plant. Kumamoto Med. J. 15:29-37.

Norton, T. R. 1975. Metabolism of toxic substances. <u>Toxicology</u>. <u>The basic science of poisons</u>. eds. L. J. Casarett, J. <u>Dovll</u>. pp. 45-132. New York: Macmillan Publishing Company.

Nowill, W. K., Stephen, C. R., Margolis, G. 1954. The chronic toxicity of trichloroethylene. Anesthesiology 15:462-465.

O'Connor, W. A. 1954. A case of trichloroethylene addiction. Brit. Med. J. 2:451-452.

Osol, A., Pratt, R. eds. 1973. United States dispensatory. 27th ed. pp. 1209-1210. Philadelphia: J. B. Lippincott Company.

Plaa, G. L., Larson, R. E. 1965. Relative nephrotoric properties of chlorinated methane, ethane, and ethylene derivatives in mice. Toxicol. Appl. Pharmacol. 7(1):37-44.

Plessner, W. 1915. Trigeminal disease as a result of trichloroethylene poisoning. Neurol. Zentralbl. 34:916-917.

Powell, J. F. 1945. Trichloroethylene: absorption, elimination, and metabolism. Brit. J. Ind. Med. 2:142-145.

Powell, J. F. 1947. The solubility or distribution coefficient of trickloroethylene in water, whole blood, and plasma. Brit. J. Ind. Med. 4:233-236.